

# A British Medical Association Lecture

ON

## DIET AND DISEASE,

*WITH SPECIAL REFERENCE TO THE TEETH, LUNGS,  
AND PRE-NATAL FEEDING.\**

EIGHTEEN months ago I delivered a British Medical Association Lecture at Bradford<sup>1a</sup> in which I discussed the subject I was actively investigating at the time—namely, the part played by modern dietary in the production of disease as found in this country. I should not have chosen the same subject for this evening's discourse were it not that it is still being intensively investigated all over the world, with results that only emphasize its importance.

Modern experimental work on animals, backed up by clinical observation, has brought to light the fact that the dietary of the people of this country is defective in two important respects: (1) that it includes too little of the substances which contain fat-soluble vitamins, and (2) that it contains relatively too much cereal. Each of these dietetic mistakes tends, among other things, to bring about a certain pathological defect of structure and function in the body, so that their combined influence in this respect is very great and produces widespread disability.<sup>1a, b, c</sup> For instance, one of the fundamental effects of a fat-soluble vitamin is to stimulate calcification of bones and teeth, while, on the other hand, excess of cereal in the diet interferes with the calcification of these organs. It can be imagined, therefore, how potent must be the destructive action on developing bones and teeth of a diet deficient in calcifying vitamin and containing an excess of cereal. The meagreness of the sunshine in this country and its poor quality so far as ultra-violet radiations are concerned only serve to make matters worse; for, arising from the observations of Huldschinsky<sup>2</sup> on the effect of ultra-violet radiations on the calcification of bones of rachitic children, we now know that deficiency of antirachitic vitamin in the diet can be made up to some extent by exposure of the body to ultra-violet light, while I have shown elsewhere that excess of cereal in the diet, which interferes with bone calcification, can also be antagonized by exposure to these rays.<sup>1c</sup>

To-night I propose to return once more to the same subject, and, by other illustrations of the action of these

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dietetic factors on the body, try to drive home, not only how widespread are the diseases for which these specific dietetic defects are responsible, but also show that it is only by feeding in such a way as to avoid these defects that success can be obtained in the control and elimination of much illness.

#### TEETH.

I propose, first of all, to deal with the subject of the teeth, and to provide some evidence obtained by May Mellanby<sup>3a-e</sup> which suggests strongly that much of the dental defect of this country is due to the dietetic mistakes mentioned above. Incidentally the results also indicate the means for combating this serious state of affairs.

It will be clear that if the fundamental cause of dental defect is due to the fact that the dietary of the people in this country is exceptionally low in calcifying properties, then the teeth ought to be badly formed. The test can be made at once. Before, however, dealing with the point, it may be well to state that the ordinary accepted teaching by dental authorities of the structure of, for instance, children's teeth in this country is that, on the whole, they are well formed, only something of the order of 2 to 3 per cent. being recognized as defective in structure. This, if true, would at once disprove the suggestion made above as to the cause of dental defect. But is it true? The 2 to 3 per cent. of defect observed in the deciduous teeth of children are cases of gross hypoplasia of the enamel, smaller abnormalities being passed as normal. It was only when teeth were examined microscopically after being ground down to thin sections that the real structure of the teeth was appreciated and correlated with the naked-eye appearance of the enamel. When this was done it was at once evident that a large proportion of human deciduous teeth were badly formed—not the 3 per cent. suggested, but something like 80 to 90 per cent.<sup>3b</sup> The following table sums up the results as to the structure of children's temporary teeth after being ground down and examined microscopically.

TABLE I.—*Relation between Structure and Caries in the Teeth of Children.*

Type of Tooth.	Number Examined.	Good Structure.		Defective Structure.	
		No Caries.	Caries.	No Caries.	Caries.
Incisors ...	100	58	11	1	30
Canines ...	70	5	0	25	40
Molars ...	466	2	11	2	451
Total ...	636	65	22	28	521

The different ratios of abnormal to normal teeth in the various types is interesting. In the case of the molars, for

instance, only 13 out of 466 teeth examined were well formed, while in the case of the incisors no less than 69 per cent. were of good structure. It will be also noticed that there was some correlation between the structure of the teeth and caries, for out of the 636 teeth examined 521 were of defective structure and carious, while 65 were of sound structure and non-carious. That is to say, 586 of these 636 teeth, or 92.14 per cent., were in agreement with the hypothesis that a sound tooth is less liable to caries and an imperfectly formed tooth more likely to be carious. Of the 636 teeth, 50, or 7.86 per cent., were in a condition opposed to this generalization. This subject has been discussed by May Mellanby elsewhere,<sup>3d</sup> and it was pointed out that the exceptions could be explained by the fact, which can also be demonstrated experimentally, that even after eruption the reaction of the teeth to harmful stimuli can be varied by the same dietetic influences which control the formation of the teeth. Thus a badly formed tooth can be made to resist more potently if the diet is made good, whereas the resistance of a well formed tooth is lowered by a defective diet.

The point I wish to make now, however, is that a very large percentage of human deciduous teeth are badly formed, and not the 2 or 3 per cent. as usually stated. In Figs. 1 and 2 can be seen cross-sections of two molars of children: Fig. 1 is a photograph of a perfectly formed molar tooth—a rarity nowadays in this country; the structure of the tooth in Fig. 2 is obviously very imperfect, and a small spot of caries is evident, yet this tooth was described by a dental surgeon as well formed judging by its external macroscopic appearance.

It is clear that children's deciduous teeth in this country are not only very susceptible to caries, but that they are very defective in structure.

The question now arises, Can the structure of teeth be controlled experimentally by diet during development, and, if so, are the dietetic factors controlling the formation of teeth of the same nature in children as in experimental animals? It is now possible, according to the experimental work of May Mellanby, to produce any degree of perfection or imperfection in the structure of the teeth of dogs by means of the diet eaten during the development of the teeth. The most important variables of the diet that are altered to bring about these differences include (1) the amount of fat-soluble vitamin,<sup>3a</sup> (2) the variation in the amount and type of cereal eaten,<sup>3c</sup> and (3) exposure of the animal, or in some cases the food, to a source of ultra-violet radiation.<sup>3e</sup> The more deficient the diet is in the calcifying vitamin, the more it consists of cereal, especially oatmeal, and the less the animal is exposed to ultra-violet radiations, the worse formed will be the teeth. On the other hand, the more the fat-soluble vitamin and the less the cereal eaten, and the greater the exposure of the animal to ultra-violet radiations, the better formed will be the teeth. These facts are demonstrated in the

illustrations (Figs. 3, 4, and 5), which are a few examples showing the effect of these dietetic and environmental factors in dental structure. For instance, the difference between the teeth and jaws in Fig. 3 is simply due to the fact that the diet of A contained some cod-liver oil, which is a rich source of calcifying vitamin, the diet of B contained a corresponding quantity of butter with a smaller vitamin content, whereas the diet of C was very deficient in this vitamin, as linseed oil formed the fat content of its diet. Except for these differences everything in the diet and environment of these three puppies, who were members of the same litter, was constant.

The destructive effect of cereals on teeth formation, and especially that of oatmeal, is seen in Fig. 4. Except in the case of A, the diets of these animals, though deficient in fat-soluble vitamins, were constant in this respect, and the variable tested was the cereal. It will be seen that when oatmeal was the cereal eaten (Fig. 4, B) the teeth were very badly formed. The abnormality was least when white flour was eaten (Fig. 4, C), and rather worse when wheat germ was substituted for 10 per cent. of the white flour (Fig. 4, D). Even the potent action of oatmeal, however, was completely antagonized by 10 c.cm. of cod-liver oil eaten daily by the dog whose jaw is represented in Fig. 4, A. The diets of the animals whose teeth are represented in Fig. 4, A and B, both contained oatmeal and were identical in other respects except that 10 c.cm. of cod-liver oil daily was eaten by A, and 10 c.cm. of olive oil by B. The tooth illustrated in Fig. 4, A, is perfect in structure.

The effect on the structure of the teeth of exposing an animal to a source of ultra-violet radiations can be seen in Fig. 5 (A and B). Both of the animals whose jaws are represented in Fig. 5 were brought up on identical diets deficient in the calcifying vitamin, and lived under the same conditions, the only difference being that one (Fig. 5, B) was exposed thrice weekly for twenty minutes to the rays of a mercury vapour lamp. The improvement in the calcification of the teeth produced thereby is obvious.

I have now shown you some evidence which indicates (1) that the deciduous teeth of children are for the most part badly formed, and (2) that the structure of dogs' teeth can be controlled at will by varying certain specific factors of diet and environment. The question arises, How do these facts bear upon the widespread scourge of caries in the teeth of children? One method of answering the question would be to feed children from birth along the lines which animal experiments have indicated as resulting in perfect tooth formation, determining the amount of caries in the temporary dentition, and after the shedding of these teeth from the sixth year onwards grinding them down and examining microscopically. A number of children are being brought up on a diet which, from birth onwards, includes milk, egg-yolk, and cod-liver oil, and up to the present, so far as is known, caries has not appeared in the

teeth of these children, but the oldest is now only  $5\frac{1}{2}$  years of age, so the test is still in its early stages.

In order to attack the point in a direct fashion with the idea of seeing whether the results obtained with animals could have any bearing on the teeth of children after eruption and after being fully formed, the following preliminary investigation was made by May Mellanby, C. Lee Pattison, and J. W. Proud.<sup>4</sup> A number of children in an institution were placed on diets which, according to animal experiments, varied in their effect on calcification. Group A were given a diet which contained much milk and some cod-liver oil, less cereal (none of it oatmeal), in addition to other food-stuffs. The diet of Group B contained less milk, more cereal (including oatmeal); while in the case of Group C an intermediate diet from the point of view of calcification, and one which was usually the standard diet of the institution, was given. The children were arranged in groups so that the average age of each group was about the same. Before the diets were started the mouth of each child was carefully charted, the amount and type of hypoplasia, the carious points, their extent and degree of hardness, the missing teeth, etc., being noted. After seven and a half to eight and a half months of the diets the condition of the teeth was again charted, and the following results were obtained.

TABLE II.—*Effect of Diets A, B, and C on the Initiation and Spread of Caries in Children.*

Diet.	Main Dietetic Difference.	New Points of Caries.	No. of Children in Group.	Average New Carious Points per Child.
A	Abundant calcifying vitamin and calcium	13	9	1.4
B	Poor in calcifying vitamin: less calcium, more cereal, especially oatmeal	51	10	5.1
C	Intermediate between A and B	38	13	2.9

It will be noticed that there was nearly four times as much new caries per child on Diet B as in the Diet A group. Since all other conditions of hygiene and mode of living were constant, it is probable that the differences in diet were responsible for the changes in the teeth of the children. If this be true, then the results observed experimentally on the teeth of dogs can be applied to children. The numbers of children observed in this investigation were small; a bigger investigation of a similar nature is necessary before the results can be regarded as definitely established. On the other hand, the amount of difference of developing caries in the various groups seems too big to be explained by inaccuracies of observation and chance.

To sum up, the experimental work demonstrating the conditions of diet which bring about the production of perfect and imperfect teeth in dogs, taken in conjunction

with the investigation on children outlined above, makes it almost certain—

(1) that the widespread development of caries in children's teeth is primarily a problem of defective feeding which results in imperfect formation of their teeth;

(2) that the dietetic factors which result in good and bad formation of teeth also confer upon or take away from the erupted teeth of children the power of resistance to the carious process;

(3) that foods containing fat-soluble vitamins, such as milk, egg-yolk, butter, animal and fish fats, and especially cod-liver oil, bring about the formation of good teeth, while cereals, and especially oatmeal, in the absence of calcifying vitamin, bring about the formation of defectively calcified teeth.

#### THE INFLUENCE OF THE MATERNAL DIET DURING PREGNANCY ON THE SUSCEPTIBILITY OF THE OFFSPRING TO DISEASE.

It would be a generally accepted proposition that the feeding and nutritional condition of a mother during pregnancy modifies the metabolic changes and consequently the well-being of the offspring. If it were only a question of insufficient nourishment supplied to the mother, evidence on this point would be difficult to obtain, for the ability possessed by the maternal organism of sacrificing her tissues for the supply of the fundamental nutriment of the developing foetus is certainly very great. There is, however, better evidence that the malnourished as opposed to the starved maternal organism transmits undesirable weakness and tendencies to pathological change to its offspring.

In the case of rickets, for instance, the maternal factor has appeared so important to some that heredity has even been advocated as the prime cause of this disease. This view has not, however, received much support. The congenital influence which has been stressed by Kassovitz is supported by such facts as the special tendency of premature babies and of twins to develop rickets. It is true, as Schmorl<sup>6</sup> pointed out, that newborn infants do not show rachitic changes of bone, but it is not improbable that the osteoporotic condition as evident in the craniotabes seen in infants soon after birth is a closely related state and may be due to malnourishment of the mother.

Experimentally Korenchevsky and Carr<sup>7</sup> showed that, in the case of rats, rachitic changes of the bones could be produced more rapidly, and more certainly, if the mother during pregnancy, as well as the offspring, were fed on diets deficient in antirachitic vitamin and calcium. Hess and Weinstock<sup>5</sup> have also studied this problem and found that, although improving the diet of mothers during pregnancy and lactation mitigated the development of rickets in infants, it did not prevent it.

In the experiments now to be described the mothers

during pregnancy were certainly not insufficiently fed. From the point of view of energy-bearing dietetic constituents the diets were not greatly dissimilar. Even as regards many of the actual foods comprising the respective diets they were identical in kind and in amount eaten. On the other hand, specific differences were introduced of such a nature that one bitch (A) received a diet which, from earlier work, would be expected to result in good health, while the second (B) received a diet which experience had taught would lead to malnutrition. The diets were as follows:

<i>Bitch A.</i>	<i>Bitch B.</i>
Bread (white flour), 150 to 200 grams.	Oatmeal, 100 to 150 grams.
Cod-liver oil, 10 to 20 c.cm.	Olive oil, 10 to 20 c.cm.
Separated milk, 400 reduced to 100 c.cm.	Separated milk, 400 reduced to 100 c.cm.
Meat, 100 grams.	Meat, 30 to 100 grams.
Yeast, 15 grams.	Yeast, 15 grams.

These animals lived throughout the experimental period under identical conditions and became pregnant at approximately the same time, the father being the same in each case. The diets were started in February, 1923, and continued throughout pregnancy, which ended on June 6th and 11th respectively, and during the period of lactation, which lasted until July 23rd and 28th, when the progeny in each case were removed from the mothers and lived separately.

After weaning, the diets were so arranged that two puppies (one puppy from each litter) were given the same food. The following table illustrates the conditions of the experiments. All puppies received the same amount of lean meat, separated milk, orange juice, salt, yeast, together with the special substances indicated in the table for each puppy.

TABLE III.

No. of Puppy.	Diet Variables.	Mother.	Father.	X-Ray Result.
698 } 709 }	Oatmeal, linseed oil, and 0.5 gram calcium carbonate	A B	R R	Nearly normal. Moderately bad rickets.
699 } 706 }	Oatmeal and linseed oil	A B	R R	Slight rickets (Fig. 6, A). Very bad rickets (Fig. 6, B).
700 } 704 }	Oatmeal and cod-liver oil	A B	R R	Normal bones. Normal bones.
701 } 708 }	White flour and cod-liver oil	A B	R R	Normal bones. Normal bones.
703 } 707 }	Oatmeal and cod-liver oil, heated and oxygenated 72 hours	A B	R R	Slight rickets (Fig. 7, ). Bad rickets (Fig. 7, B).

The only results that need be referred to here are those of the dogs on diets which would be expected to interfere most severely with bone calcification. These are 698 and 709 (oatmeal, linseed oil, and calcium carbonate), 699 and 706 (oatmeal and linseed oil), and 703 and 707 (oatmeal and cod-liver oil, heated and oxygenated seventy-two hours). Since there is little or no calcifying vitamin in linseed oil, and that present in cod-liver oil is destroyed by seventy-two hours' heating and oxygenation, none of these diets contained the necessary amount of vitamin. The results obtained in the case of 699 and 706 (oatmeal and linseed oil) can be seen in Fig. 6 (A and B), where the radiographs after ten weeks of the diet are shown. It will be seen that 706 (defectively fed mother—Fig. 6, B) has advanced rickets in the radiograph, whereas the radiograph of 699 (well fed mother—Fig. 6, A) shows only slight rickets at this time. Since the only difference in the life-history of these two dogs is that the mother of one (706) was defectively fed during pregnancy and lactation, while the mother of 699 had a good, strongly calcifying diet, it is probable that this is responsible for the increased susceptibility of 706 to develop rickets as compared with 699.

A similar result was obtained with 703 and 707, where heated and oxygenated cod-liver oil was the fat eaten by both. It will be obvious in Fig. 7, A and B, that 707 (defectively fed mother—Fig. 7, B) has developed more severe rickets than 703 (well fed mother—Fig. 7, A), and the reason for this is probably the same as in the case of the preceding pair. Animals 698 and 709 (radiographs not shown) reacted in the same way: 709, having the defectively fed mother, developed much more severe rickets than 698 (well fed mother).

It was a matter of interest to know how long the influence of the defective diet of the mother would be evident in the offspring, and more information was obtained from other puppies in these litters in the following way. Animals 701 (well fed mother) and 708 (badly fed mother), after weaning, were given a good diet containing an abundance of antirachitic vitamin. This continued for four months, and by this time the puppies were in excellent condition and about 6 months old. The diet of each was then changed to a defective one by the substitution of oatmeal for white flour and olive oil for cod-liver oil. After six weeks of this defective diet it will be seen that the bones of 708 (defectively fed mother—Fig. 8, B) and of 701 (well fed mother—Fig. 8, A) were still practically normal. These animals were now  $7\frac{1}{2}$  months old and well grown dogs. However, after three months more of these defective diets, when they were  $10\frac{1}{2}$  months old, there was a great difference between these two animals, which is obvious in the radiographs and photographs of these dogs (Fig. 8, C and D, and Fig. 9, E and F). It will be seen that 708 (Fig. 8, D, and Fig. 9, F) is very rachitic in appearance as compared with 701 (Fig. 8, C, and Fig. 9, E).

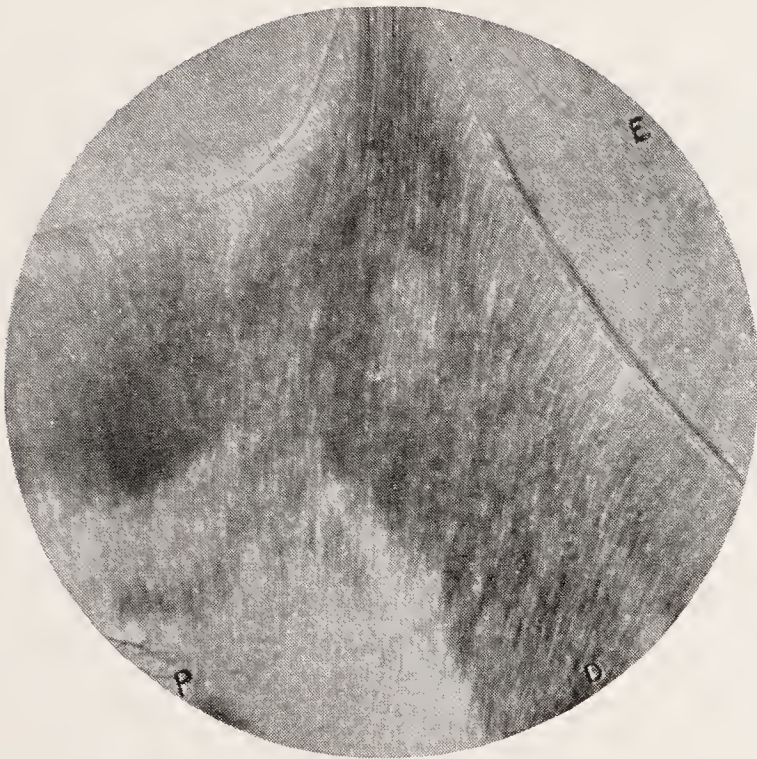


FIG. 1.—Photomicrograph of ground section of a perfectly formed human deciduous molar. Rarely found. (May Mellanby.)

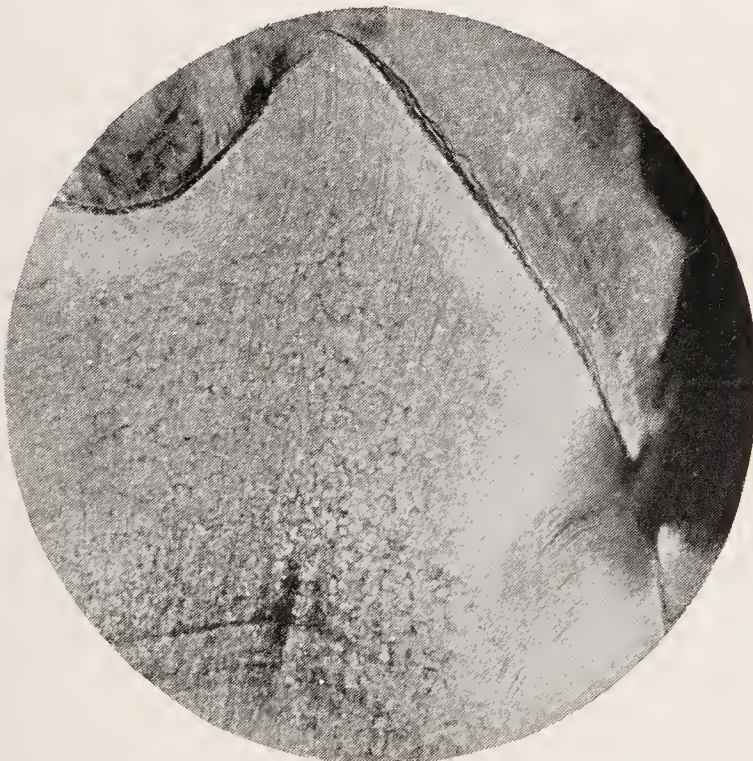


FIG. 2.—Photomicrograph of human deciduous molar. Note defect in structure of dentine. A typical specimen as ordinarily found in this country. (May Mellanby.)

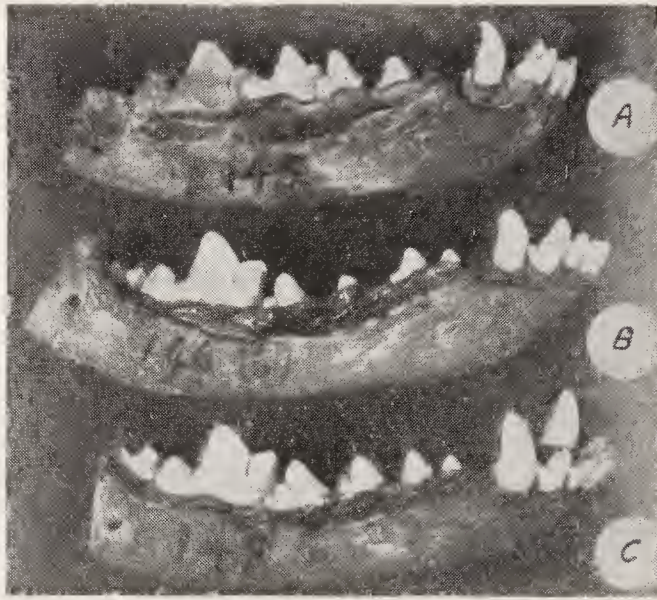


FIG. 3 (A, B, and C).—The effect of the calcifying vitamin. The jaws of three puppies of the same litter brought up on the same diets except that A contained 10 c.cm. of linseed oil daily, B contained 10 grams of butter daily, and C contained 10 c.cm. of cod-liver oil daily. Note the perfect formation of the teeth of C and the imperfectly formed teeth in A. (May Mellanby.)



FIG. 5 (A and B).—The effect of ultra-violet radiations. Photographs of the lower jaws of two puppies brought up on the same diet deficient in calcifying vitamin and living under the same conditions. Puppy A only was exposed thrice weekly for twenty minutes to the radiations of a mercury vapour lamp. Note the better formed teeth of A as compared with B. (May Mellanby.)



FIG. 4 (A, B, C, and D).—The effect of different cereals. Photomicrographs of ground sections of molar teeth of four puppies of the same litter. The diets of B, C, and D were deficient in antirachitic vitamin and were identical except that B contained oatmeal as cereal, C contained white flour as cereal, and D contained white flour and wheat germ (10 per cent.) as cereal. The diet of A was identical with that of B—that is, it contained oatmeal as cereal, but olive oil in Diet B was replaced by 10 c.cm. of cod-liver oil, which completely antagonized the bad effect of the oatmeal. Note how defective is the dentine in B (oatmeal), also that wheat germ has made the teeth of D worse than C (white flour). (May Mellanby.)

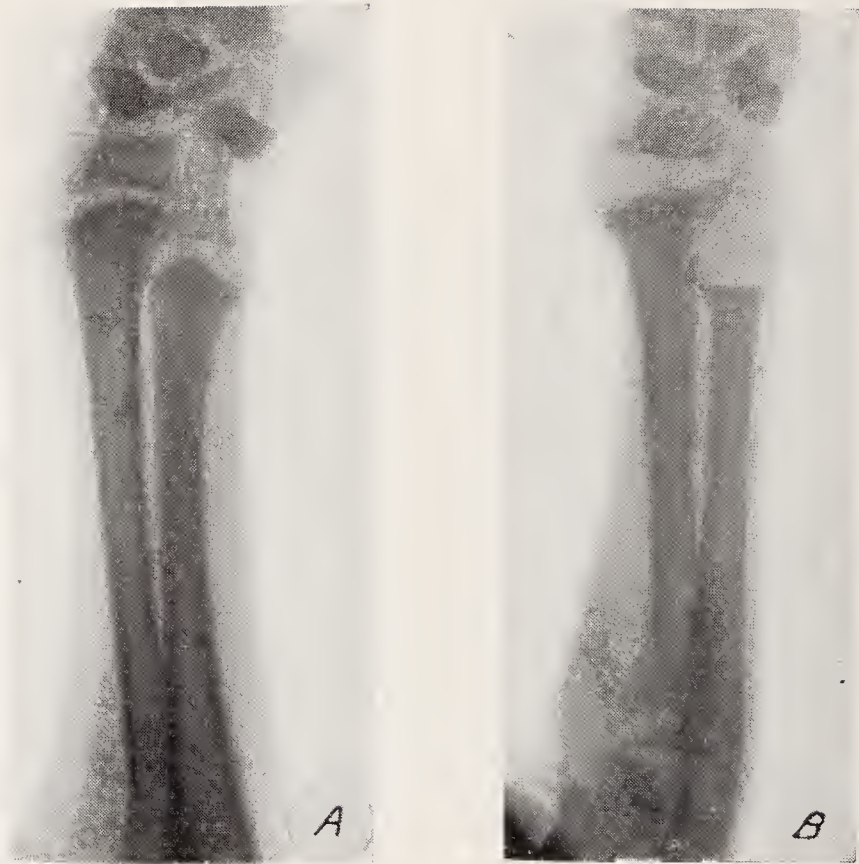


FIG. 6 (A and B).—Radiographs of wrists of puppies (699 and 706) taken after ten weeks of the same experimental diet. A, during pregnancy and lactation the mother of 699 had been well fed—that is, received abundant antirachitic vitamin. B, during pregnancy and lactation the mother of 706 had been fed on defective diet—that is, deficient in antirachitic vitamin.



FIG. 7 (A and B).—Radiographs of wrists of puppies (703 and 707) taken after ten weeks of the same experimental diet. The mother of A (703) had been on a diet deficient in antirachitic vitamin during pregnancy and lactation, while the mother of B (707) had had a diet good in this and other respects during the same period.

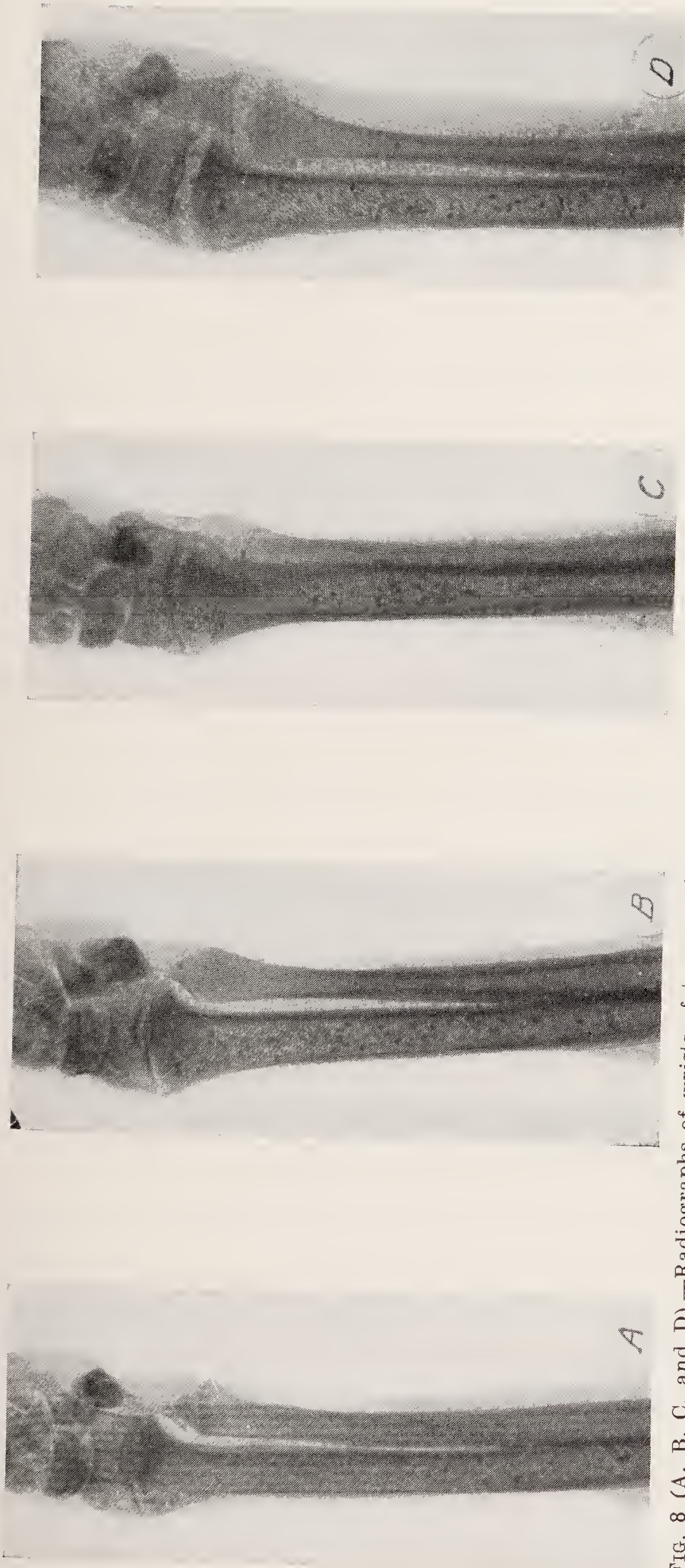


FIG. 8 (A, B, C, and D).—Radiographs of wrists of two puppies (701 and 708). The mother of 701 (A and C) was well fed during pregnancy and lactation. The mother of 708 (B and D) during pregnancy and lactation had a diet deficient in antirachitic vitamin, and containing, among other things, oatmeal. After weaning the diets of 701 and 708 were always identical; they were well fed from 6 weeks to 6 months old; they were then put on the same rickets-producing diet. Radiographs A (701) and B (708) after six weeks of the defective diet; Radiographs C (701) and D (708) after eighteen weeks of the defective diet. See also Fig. 9, in text.

The rachitic changes in 708 (Fig. 8, D, and Fig. 9, F) are comparable to those of late rickets seen occasionally in adolescents.

The greater resistance of 701 to the development of late rickets and the susceptibility of 708 to this disorder must have been due to the difference of the maternal feeding during pregnancy and lactation; for, since weaning at the age of 6 weeks, the diets of each and all the other conditions of life have been identical. It seems clear, then, that the influence of a defective diet given during pregnancy and lactation is not only evident in the offspring during early life, but that, even after a prolonged period of perfect feeding, the tendency to the development of defect is still obvious in the case of dogs whose mothers have a bad diet. It is surprising that the defective diet effect should be so lasting, and that it should be so difficult to overcome by a period of excellent treatment of the puppy.

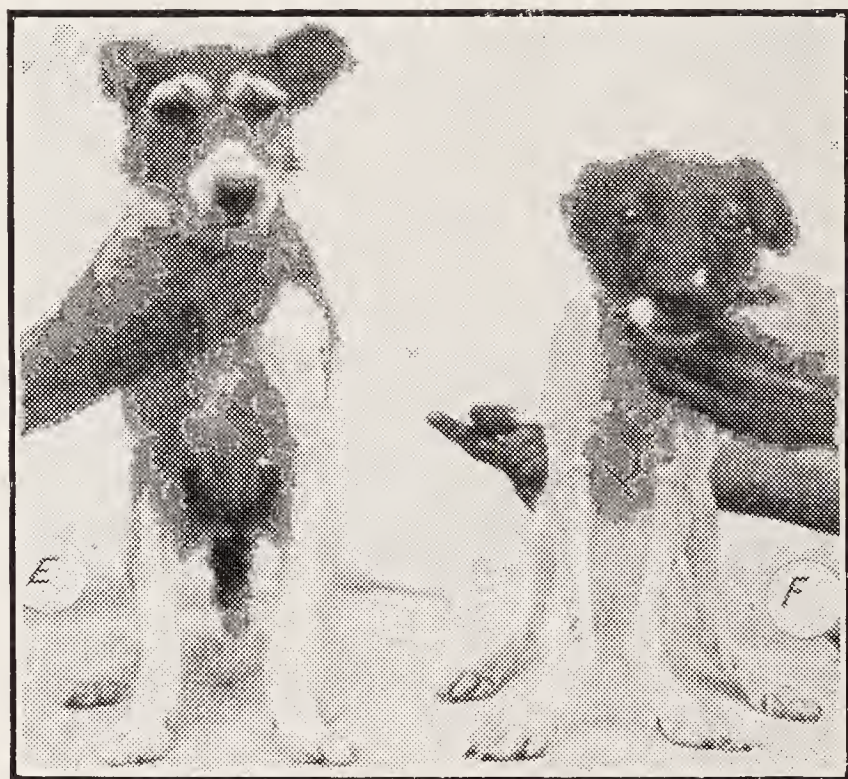


FIG. 9.—Photographs of two puppies—E (701) and F (708)—after eighteen weeks of defective diet. Animal 708 (badly fed mother) has developed severe “adult rickets” as compared with 701 (well fed mother). For radiographs and details as to diet see Fig 8 in special plate.

### *Summary.*

If bitches are fed during pregnancy on diets which, in the case of puppies, will lead to rickets, then the offspring have a greater tendency to develop this disease. This tendency in the young is not removed by a period of good diet, but may become evident again at a later period of defective feeding.

## THE RELATION OF DIET TO SUSCEPTIBILITY TO INFECTIONS OF THE RESPIRATORY TRACT.

Attention has been drawn by many recent workers on dietetics to the increased susceptibility of animals to infection as the result of diets which, while good as regards their protein, fat, carbohydrate, and energy content, are defective in quality. McCarrison,<sup>8</sup> for instance, has described the frequency of a catarrhal condition of the intestine, especially in the form of colitis, found in animals whose diet was deficient in vitamin B. In the course of my own work I commented on the readiness with which dogs succumb to distemper and the great intensity of mange when these diseases appear in animals whose diets are deficient in vitamin A.<sup>1b</sup> Drummond<sup>9</sup> has described a lowering of resistance of adult rats to bacterial infection, manifesting itself sometimes in the form of an inflammatory condition of the lungs, when they were fed on diets deficient in vitamin A. Cramer and Kingsbury<sup>10</sup> have also called attention to the mortality of rats from bronchopneumonia when these animals are kept sufficiently long on diets free from vitamin A. They consider that the atrophy of mucus-secreting cells in the mucous membrane of the trachea and larynx, observed by Mori,<sup>11</sup> and in the intestine, observed by themselves, allows the local bacterial infection of these tissues when the diet is deficient in vitamin A. These are a few of the instances in which attention has been called to this important problem. Any information on the question of the relation of diet to resistance to infection, especially as it concerns the respiratory tract, seems to me to be of such great practical importance that I think it necessary to give my own experience on the subject.

At one period in the course of my experimental investigations on dogs, the work was greatly hampered by the development of an inflammatory condition of the lungs. Another condition of the lungs found *post mortem* in some of the dogs was that of local collapse, especially along the margins of the upper lobes of the lungs. This was often associated with patches of emphysema in other parts of the lungs. This condition, as a general rule, was not accompanied by congestion. It was usually found in dogs which, on account of their diet, had become either very lethargic or had developed muscular weakness or both.

Many of the experiments have been made with the object of elucidating the effect of diet on bone formation, so that the number of animals developing bone defect has been large. This, no doubt, accounts for the frequency with which muscular weakness and the accompanying condition of local lung collapse or atelectasis occur in these dogs.

Lately I have examined microscopically many of the lungs seen to be abnormal at the *post-mortem* examination made at the end of each experiment, and found the inflammatory conditions in all cases represented varying degrees of bronchopneumonia. It then seemed worth while

to analyse the results and see in what way, if any, the incidence of the bronchopneumonia was related to the diet. I will briefly tabulate these results and then discuss them.

TABLE IV.—*Lung Condition found at Post-mortem Examination of a Series of 330 Dogs in Relation to Diet.*

Condition of Lungs.	Diets as regards Vitamin A Content.				
	Vegetable Fat Vitamin A Deficient.	Butter + Vitamin A	Butter, Vitamin A Destroyed by Heat.	Cod-liver Oil + Vitamin A.	Cod-liver Oil, Vitamin A Destroyed by Heat.
Normal ... ..	155	55	11	24	3
Broncho-pneumonia	43	0	4	0	1
Local collapse ...	23	9	2	0	0
Total ... ..	221	64	17	24	4

These results indicate a close relationship between the fat-soluble vitamin content of the diet and the susceptibility of the animal to develop an inflammatory condition of the lungs. All the cases of bronchopneumonia were found in dogs whose diets were deficient in fat-soluble vitamin, and no bronchopneumonia developed when the diet contained either butter or cod-liver oil. Except for the variable amount of fat-soluble vitamin, the diets of these animals would formerly have been considered good—that is to say, they contained an abundance of protein, fat, carbohydrate, and energy. In some cases the calcium intake was only sufficient on the assumption that the diet contained a large amount of calcifying vitamin. For instance, if diets of this nature contained cod-liver oil as the fat entity, and therefore an abundance of calcifying vitamin, the calcium intake was sufficiently high to result in perfectly formed teeth and bones. In fact, it is to be doubted whether any ordinary diet can be so low in calcium content as to lead to defective calcification of the body tissues if cod-liver oil is also ingested at the same time. On the other hand, when butter is the source of fat-soluble vitamin in the diet it is essential, in order to produce well formed teeth and bones, strongly contracting muscles, and good general activity, that the diet should also be richer in calcium. The reason for this is probably that butter contains a much smaller amount of antirachitic vitamin than cod-liver oil, so that the butter effect on calcification is best seen when there is plenty of calcium in the diet. In the above table it will be seen that local collapse of lung tissue was found in nine cases in which butter was the fat eaten. If the calcium of the diets in these particular cases had been higher, the musculature of the animals would have been stronger, the

animals would have been more vigorous, and no local collapse of lungs would have developed. Yet none of these animals eating butter, although some were abnormal in other ways, even as regards the lungs, developed bronchopneumonia. Although, therefore, it is certain that there is an intimate relation between fat-soluble vitamin in the diet and calcium, especially as regards the structure and function of bones, teeth, and muscle, it seems possible to deduce, on the basis of the above statistics, that this vitamin alone confers an increased power of resistance to lung infection even under conditions when bone and muscle structure is defective.

It is necessary, in discussing this question as to the relationship of diet and the resistance of the body to inflammatory conditions of the respiratory tract, to emphasize that, whatever truth there may be in the suggestion that fat-soluble vitamin aids in the defensive mechanism, it is only one point of a more complicated story. Whether there was any special organism which invaded the lungs of these dogs is not yet known, as, up to the present, the subject has not been studied in detail. There are certainly other points of crucial importance in the etiology of bronchopneumonia, but what these are cannot be stated definitely. In some cases, at least, the bronchopneumonia developed in animals which not only had the diet defect described above, but also had been taken out of their indoor kennels into the open air, where it was usually cold and windy and often wet, in order that their running powers should be determined. The opportunity, in fact, was presented to them of catching a "chill." The dogs on the diets containing vitamin A were also placed under the same conditions, but their resistance was apparently sufficient to make the low temperature of the external conditions of no account. It is impossible to state that this change of environment, which lasted only a few minutes, was always a factor in the development of the inflammatory condition of the respiratory tract, but it may have been a causative agent in many cases.

Another condition which commonly develops in puppies when feeding on diets containing excess of cereal and a deficiency of fat-soluble vitamin is diarrhoea. In animals which feed on such a diet for a long enough period diarrhoea generally develops, but this may only happen after severe defect of bone formation is present. On the other hand, diarrhoea may appear soon after the diet begins and before there is obvious bone deformity. It has seemed, although not proved, that the better the puppies are fed during the pre-experimental period, the longer time will elapse before the defective diets are accompanied by diarrhoea. This statement is definitely true of rickets both as regards the pre-natal and post-natal feeding of the mother, and it probably applies equally to the development of catarrhal conditions of the alimentary tract. It may also explain the variable susceptibility of young animals to catarrhal and inflammatory conditions of the respiratory tract.

THE POSSIBLE BEARING OF THE ABOVE RESULTS ON  
THE "CATARRHAL" CHILD.

The observations described above, dealing with the effect of feeding the maternal organism during pregnancy and lactation on the susceptibility of the young to develop rickets, together with the results showing the altered resistance under similar dietetic conditions to inflammation of the respiratory passage, have impressed upon me the possibility that the so-called "catarrhal" child is probably a product of defective feeding of the mother during pregnancy and lactation. There is general agreement among clinical workers that there is some common factor in the etiology of the diseases which result in the "catarrhal" child, the rachitic child, and the child with enlarged tonsils. So far as my own animal experiments are concerned, I have got no evidence that enlarged tonsils of the type seen so commonly in children are produced by dietetic defect, but this may be because dogs do not develop the condition at all, or because I do not keep them long enough on bad diets. Clinically, however, it would probably be agreed that chronic catarrh of the respiratory passages of children, tendency to bronchopneumonia, rickets, attacks of diarrhoea, and, later, enlarged tonsils, are intimately related.

The catarrhal condition in children may develop at any time, but often it appears in the first few weeks of life and before post-natal conditions in themselves could be accounted responsible for the absence of all resistance to this type of infection. It seems to me that such cases can probably be explained on the basis of defective feeding of the mother during pregnancy, and the defects are probably of the type indicated by some of the experimental work on animals described in this lecture—namely, a deficiency in the diet of foods containing fat-soluble vitamin, such as milk, eggs, butter, cheese, animal and fish fat, and a relative excess of cereals such as bread, oatmeal, and rice, and other foodstuffs deficient in vitamin A. The basis of this suggestion I have given above, and may be summarized as follows:

(1) It has been shown experimentally that these defects in the maternal diet increase the tendency of the offspring to develop rickets.

(2) The same defects in the diet seem, on the basis of the statistics supplied above, to increase the susceptibility of young animals to bronchopneumonia and inflammatory conditions of the respiratory tract, and, in general, to result in puppies of lowered vitality.

(3) It is well known that the catarrhal child may develop this condition shortly after birth, and that it has a great tendency to become rachitic and to develop bronchopneumonia.

(4) Puppies which develop rickets when feeding on these experimental diets frequently develop a catarrhal condition of the alimentary tract sooner or later, the time seeming to depend partly upon the kind of feeding of the mother and the puppy in the pre-experimental days.

Not only is there some experimental support for the suggestion, but experience shows that the dietetic defects described are those most commonly met with in human feeding. It is therefore probable that these defects of diet of women during pregnancy and lactation are responsible for some, and possibly much, of the illness and mortality of young infants. The new teachings of diet have been applied to some extent to the feeding of children, and this is no doubt partly responsible for the decrease in infant mortality during recent years, at a time when overcrowding and some other hygienic defects are as bad as, or even worse than, ever; but it is necessary to extend the teaching to the problem of maternal feeding. This would probably show its first effect by reducing the infant mortality of children under 1 month of age, and, if the foregoing suggestions and experimental results are true, would result in great improvement in the physique of children. It would increase the resistance of infants to those infections which produce catarrhal conditions of the respiratory and alimentary tracts and all the other sequelae so generally recognized as likely to follow. It would certainly result in a better grown and less rickety type of child, and would do something also to improve the structure of the teeth and thereby to reduce the appalling amount of caries in the teeth of children. It would probably also bring about improvement in the general health of the pregnant woman, and do away with some of the unfortunate experiences to which she is liable.

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